



Prevalence and characteristics of CAAs in the black sea region

CAAs in the black sea region

Osman Kayapinar¹, Ahmet Egemen Sayin², Adnan Kaya¹, Cem Ozde¹, Muhammed Keskin³

¹Cardiology, Duzce University Medical School, Duzce,

²Cardiology, Nusaybin State Hospital, Mardin,

³Cardiology, Sultan Abulhamid Han Training Hospital, Istanbul, Turkey

Abstract

Aim: To date there has been no data about the prevalence of coronary artery anomaly (CAA) in the Turkish population of the Black Sea Region who underwent trans-radial coronary angiography. We aimed to determine the frequency and characteristics of CAA in our patients. Material and Method: All the coronary angiographies performed from September 2015 to September 2016 in our hospital were reviewed. Demographic characteristics and laboratory parameters of patients were reviewed retrospectively from the patients' data set. A total of 1617 patient were included in our study. Results: CAAs were found in 73 patients (4.51%), of whom 41 (56.16%) had intrinsic coronary artery anatomy. Twenty-two (30.13%) patients had anomaly of origination and course and 10 (13.69%) patients had anomaly of coronary artery termination. The mean age was 59.35 ± 11.86 in the study group and 60.11 ± 6.61 in the control group. Myocardial bridge was the most common anomaly in our study with a prevalence of 2.16%. Absent LMCA was the second most common anomaly in our study with a prevalence of 0.80% and coronary artery fistula was third with a prevalence of 0.61%. Discussion: We found the prevalence of CAAs among the Turkish population of the Black Sea Region to be similar to previously published studies from our country. To avoid misunderstandings one must know the normal anatomy of coronary vasculature, variations, and the anomalies. When coronary angioplasty or cardiac surgery is planned in patients with CAA, special attention must be paid not to harm coronary arteries in unexpected locations.

Keywords

Coronary Artery Anomaly; Trans-Radial Coronary Angiography; Black Sea Region

DOI: 10.4328/JCAM.5731 Received: 26.01.2018 Accepted: 04.03.2018 Published Online: 08.03.2018 Printed: 01.09.2018 J Clin Anal Med 2018;9(5): 369-75
Corresponding Author: Adnan Kaya, Department of Cardiology, Duzce University School of Medicine, Istanbul, Turkey.
GSM: +905324009765 E-Mail: adnankaya@gmail.com
ORCID ID: 0000-0002-9225-8353

Introduction

Coronary artery anomaly (CAA) is one of the main reasons of sudden cardiac death among young athletes. CAA is not completely understood and its importance has been neglected so far. CAA is defined as coronary arteries not fulfilling the criteria of normal coronary artery anatomy.

Angelini has proposed that 99% of the population (2 to 3 standard deviations) can be assumed to have variations within normality (normal variants) and that the remaining 1% of population (outside 2 to 3 standard deviations) could be assumed to have CAA [1]. Despite this assumption there is no real world data to support this statement. Data about the prevalence of CAA comes from post mortem autopsy series of deaths with unspecified causes and coronary angiographies of symptomatic patients. Data derived from the autopsy series showed a relatively lower incidence of up to 0.03% [2] and data derived from the coronary angiography series showed an incidence up to 5.6% [3].

Coronary arteries arise from the sinus of Valsalva (SoV) just above the aortic valve from the ascending aorta to supply the myocardium. Two main trunks of coronary arteries share normal myocardium perfusion: left main coronary artery (LMCA) and right coronary artery (RCA). LMCA arises from the left SoV and divides into left anterior descending artery (LAD) and circumflex artery (CX). However, occasionally LMCA divides into three vessels: intermediate artery (IM) additional to LAD and CX. LAD follows the anterior interventricular groove to the apex of the heart while supplying anterior and lateral of the left ventricle (LV) with diagonal branches and the septum with septal branches. CX follows the left atrioventricular groove posteriorly while supplying the lateral and free wall of LV with obtuse marginal (OM) branches and inferior wall of LV with posterior descending artery (PDA) in about 15% of patients. When IM is present, it supplies the free wall of LV where diagonal and OM could not. RCA follows the right atrioventricular groove posteriorly while supplying right ventricle (RV) with RV branches and LV inferior with PDA in about 85% of general population.

Most of the coronary anomalies permit normal myocardial development and function, resulting in a normal life expectancy. Moreover, intense athletic activity could be performed while having a coronary anomaly. However, there is a wide range of symptoms and pathologies considered to lead to sudden cardiac death that are associated with coronary anomalies. Myocardial ischemia associated clinical presentations in CAA include chest pain, sudden death, cardiomyopathy, syncope, dyspnea, ventricular fibrillation, and myocardial infarction. Volume overload, increased risk of bacterial endocarditis, increased risk of secondary aortic valve diseases, difficulties during diagnostic angiography or procedural angioplasty and increased risk of complications during cardiac surgery are the other inconveniences of coronary anomalies [3].

The purpose of this study was to determine the prevalence of origination, course, and termination anomalies of coronary arteries among Black Sea Region Turkish population who underwent trans-radial coronary angiography for any reason.

Material and Method

Study Design and Patient Selection

This is a single center retrospective study comprising all the patients who underwent coronary angiography from September 2015 to September 2016 in our tertiary university hospital. A total of 1899 coronary angiographies were performed during this interval. Two hundred eighty-two patients were excluded from the study according to the exclusion criteria, which were as follows:

- i). Patients under 18 years old,
- ii). Previous cardiac surgery,
- iii). Inconclusive coronary images,
- iv). Patients complicated with cardiopulmonary arrest,
- v). Patients with connective tissue disorder,
- vi). Patients with coronary anomalies associated with complex congenital heart disease,
- vii). Patients with one or more totally occluded epicardial coronary artery.
- viii). Patients who underwent coronary angiography through the trans femoral route.

Coronary Angiography Evaluation

All the coronary angiography images were evaluated by two interventional cardiologists and coronary artery anomalies were diagnosed by consensus. Patients were excluded when it was impossible to make a diagnosis. Coronary artery anomalies were defined and classified according to the classification proposed by Angelini. All the coronary angiography images were investigated for anomalous take off from aorta, abnormal course (myocardial bridges), abnormal termination (coronary fistulas), and abnormal coronary collateral arteries.

The coronary images were further evaluated for i) absence of left main coronary artery (separate ostia of LAD and CX), ii) atypical beginning of LAD (arising from right aortic sinus or RCA), iii) atypical beginning of CX (arising from right aortic sinus or RCA), and iv) atypical beginning of RCA (arising from left aortic sinus, arising from CX or presence of split RCA which could be defined as two different RCA originating from right aortic sinus or splitting of RCA into two duplicates shortly after arising from right aortic sinus).

Myocardial bridge diagnosis was made according to the presence of 30% or more systolic compression of coronary arteries that resolve in diastole. When more than one myocardial bridge is present in the same coronary artery it is accepted as a single myocardial bridge.

Coronary artery fistulas were investigated attentively for their origin, course, and termination. When there were two different anomalies (e.g., presence of MB and split RCA or coronary artery fistula and split RCA) in the same patient, it was accepted as a single patient but two different anomalies.

Three experienced interventional cardiologists performed all the coronary angiographies according to current guidelines through the right radial route.

Patient Demographic Characteristics, Laboratory Parameters, and Ethics Approval

Demographic properties and laboratory parameters of the patients were obtained from the patients' data set. All the blood samples were drawn one or two days prior to the procedure in elective patients and within 2 hours in patients with unstable

angina, non ST-elevation myocardial infarction, and ST-elevation myocardial infarction. The study was approved by the Ethics Committee of the University.

Statistic Analysis

The SPSS statistical software package (SPSS, version 19.0 for windows; SPSS Inc., Chicago, IL, USA) was used for all the statistical calculations. Continuous variables were expressed as mean ± S.D.; categorical variables were shown as percentages. Student’s t-test was used to compare continuous variables whereas chi-square was used to compare categorical variables. For all the tests, a value of p<0.05 was considered to be statistically significant.

Results

A total of 1617 consecutive patients were included in our study. Seventy-three of them (4.51%) were found to have CAA. Twenty (27.39%) of the CAA patients and 445 (28.82%) of the control patients were female. Mean age was 59.35±11.86 in the study group and 60.11±6.61 in the control group. Groups were similar in relation of cardiovascular risk factors (Hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HL), smoking, body mass index (BMI), chronic kidney disease (CKD), previous coronary artery disease (CAD), stroke, and baseline biochemistry (glucose, thyroid stimulating hormone (TSH), e-GFRC-G, creatinine) and hematological (hematocrit (HCT), white blood cell count (WBC), platelet count) factors (Table 1).

Table 1. Demographic characteristics, risk factors identification and baseline laboratory parameters of patients.			
	CAA (n=73, 4.51%)	Controls (n=1544, 95.48%)	P
Age, years	59.35±11.86	60.11±6.61	0.478
Sex (Female, (%))	20 (27.39%)	445(28.82%)	0.793
BMI, kg/m²	26.83±4.22	27.17±2.37	0.511
Hypertension, (%)	43(58.90%)	891(57.70%)	0.904
Diabetes mellitus, (%)	21(28.76%)	457(29.59%)	0.879
Hyperlipidemia, (%)	15(20.54%)	318(20.59%)	0.914
Smoking, (%)	19(26.02%)	313(20.27%)	0.234
Chronic kidney disease, (%)	6(8.21%)	117(7.57 %)	0.820
Stroke, (%)	3(4.10%)	70(4.53%)	0.865
CAD, (%)	15(20.54%)	293(18.97%)	0.738
Hematocrite, %	40,8±5,6	40,9±4,8	0.841
White Blood Cell, 10 ⁵ / µL	8,9±4,3	8,1±3,1	0.140
Platelet count, 10 ³ / µL	253±93	240±69	0.130
Glucose, mg/dL	98±48	101±39	0.755
Creatinine, mg/dL	0,78±0.10	0.81±0.07	0.228
e-GFR _{C-G}	104±13	106±6	0.850
TSH IU/dL	2,1±1.6	2.3±1.1	0.532

CAA; coronary artery anomaly, CAD: Coronary artery disease, BMI: Body mass index, TSH: thyroid stimulating hormone, eGFR_{C-G} : estimated glomerular filtration rate with Cockcroft-Gault equation

Coronary artery anomalies were classified according to the systemic anatomic classification proposed by Angelini (1). Seventy-three patients of the total 1617 patients were identified as having CAA, with a prevalence of 4.51%. Of these 73

patients, 41 (56.16%) were found to have anomaly of intrinsic coronary artery anatomy; 22 (30.13%) patients were found to have anomaly of origination and course; and 10 (13.69%) were found to have anomaly of coronary artery termination (Table 2). Myocardial bridge was the most common anomaly in our study with a prevalence of 2.16%. Absent LMCA was the second most common anomaly in our study with a prevalence of 0.80%. Prevalence of other anomalies were as follows: coronary artery fistula at 0.61%, CX arising from right anterior sinus at 0.18%, RCA arising from left anterior sinus at 0.12%, absent OM at 0.12%, woven coronary anomaly at 0.12%, LMCA arising from right anterior sinus at 0.061%, RCA arising from non coronary sinus at 0.061%, PDA arising from right anterior sinus at 0.061%, single coronary artery at 0.061%, dual LAD at 0.061%, and coronary crossing at 0.061% (**Table 3**). Four patients had more than one anomaly. One patient had both LAD and RCA fistulas, another had RCA fistula and myocardial bridge. One patient had woven coronary anomaly in OM and IM. One patient had myocardial bridge and coronary crossing. In these patients each anomaly was included in its own group separately.

Table 2. Frequencies of coronary artery anomalies according to systemic anatomic classification.		
	N	%
Anomalies of origination and course	22	30.13
Anomalies of intrinsic coronary arterial anatomy	41	56.16
Anomalies of coronary termination	10	13.69
Anomalous collateral vessels	0	0
Total	73	100

Table 3. Isolated coronary artery anomalies and the prevalences.			
Type of Coronary anomaly	No	Prevalence (%)	Anomalies (%)
Myocardial bridge	35	2.16	47.94
Absent LMCA	13	0.80	17.80
Coronary artery fistula	10	0.61	13.69
CX arising from right anterior sinus,	3	0.18	4.10
RCA arising from left anterior sinus	2	0.12	2.73
Absent OM	2	0.12	2.73
Woven coronary anomaly	2	0.12	2.73
LMCA arising from right anterior sinus,	1	0.061	1.36
RCA arising from non coronary sinus	1	0.061	1.36
PDA arising from right anterior sinus	1	0.061	1.36
Single coronary artery	1	0.061	1.36
Dual LAD	1	0.061	1.36
Coronary crossing	1	0.061	1.36
Total	73	4.51	100

LMCA: Left main coronary artery, CX: circumflex artery, RCA: right coronary artery, OM: obtus marginalis, PDA: posterior descending artery, LAD: left anterior descending artery

Discussion

The present study found that 4.51% of Turkish patients from the Black Sea Region who underwent trans-radial coronary angiography for any reason have CAAs according to Angelini’s definition (origination, course, and termination anomalies of coronary arteries). Anomaly of intrinsic coronary artery anatomy was the most frequently seen with 56.16% whereas the

frequency of anomaly of origination and course was 30.13% and the frequency of anomaly of coronary artery termination was 13.69%. The prevalence of CAAs was reported to vary from 0.2% to 8.4% in previous studies [4, 5]. Our study was in harmony with previous studies in relation to the frequency of CAA. However, our prevalence findings were higher than those in the previously conducted studies from our country [6-9]. This difference was attributed to variability of the classification of CAA from study to study. Myocardial bridge, one of the most frequently seen anomalies of coronary artery anatomy, was not accepted as an anomaly and excluded from some studies [10]. Another explanation for this discrepancy could be variability of CAAs between regions in a country and between countries.

Baseline characteristics of the patients were similar among groups in our study. The prevalence of HT was similar to previously published studies [11]. However, DM was found to be more common than in previously published studies from our country [12]. This was attributed to an older and selected population for higher risk of having CAD. Previous CAD prevalence was similar between groups. Despite contradictory ideas about the association of CAA with obstructive CAD, it is generally accepted that normal segments of coronary arteries of a person with anomalous course have a similar risk of developing obstructive coronary artery disease. Hematologic and biochemistry parameters were similar between groups in our study. Similar demographic and laboratory characteristics between groups make it difficult to predict which patients may have CAAs.

Myocardial bridge (Figure 1: A-B)

Incidentally found during a selective coronary angiography or at autopsy, CAAs exist as early as birth and are thought to have a genetic background [10, 13, 14]. The real prevalence of CAAs is not known. The range of prevalences reported in the literature may be above or under the real prevalence based on the classification of CAAs and selection bias.

Myocardial bridge, once believed to be a benign condition, is a passage of a part of an epicardial coronary artery under the myocardium that can compromise blood flow during systole. LAD is the most affected coronary artery. Location and length of the myocardial bridge, thickness of myocardium, and the degree of cardiac contractility are the main determinants of systolic compression. Sudden cardiac death [15], exercise-induced atrio-ventricular conduction block [16], stunning [17], arrhythmias [18], ventricular septal rupture [19], coronary spasm [20], ischemia and acute coronary syndromes [21] are the defined clinical presentations associated with myocardial bridge. Its prevalence varies from 1.5% to 16% in angiography series and as high as 80% in autopsy series [22, 23]. In our study the prevalence of myocardial bridge was 2.16% (n=35, study population=1617) which accounts for 47.94% (n=35, patients with CAAs=73) of CAAs. Our data is compatible with the literature. Myocardial bridge, previously seen as an harmless anatomic variant of the coronary artery course, nowadays is believed to have fatal consequences. However, the exact mechanism for this is yet to be clarified. Several pathophysiologic explanations have been suggested: i). Most of the myocardial perfusion occurs during diastole (with estimated 85% of blood flow) and less during systole (with estimated 15% of blood flow). Since

myocardial bridge is a systolic compromise in nature, one can consider that is not a significant factor to induce ischemia. However, when the heartbeats accelerate (in tachycardia states), systolic blood flow gains importance due to shortened diastolic filling time [24] and decreased coronary flow reserve (a measure of the ability to augment coronary blood flow under stress) [25]. ii). Kinking of the coronary arteries during tachycardia may cause endothelial damage due to continuous trauma in turn ending with platelet aggregation, vasospasm and acute coronary syndromes [26]. iii). Moderate-to-high laminar shear stress is the most important survival factor for endothelial cells to produce and release NO (nitric oxide). Due to distorted normal laminar blood flow pattern, NO production and bioavailability decrease and the coronary artery is exposed to ET-1 (endothelin-1) effect with a reduction or complete loss of NO to counteract it [27]. After all, myocardial bridge leads interstitial fibrosis, replacement fibrosis, contraction band necrosis, increased vascular density in the affected area and leads to variable clinical conditions.

Separate origin of LAD and CX from the left sinus of Valsalva (absent left main trunk) (Figure 1: C-D).

One of the milestone studies about incidence and characteristics of CAAs was conducted by Yamanaka O and Hobbs RE 1990 [10]. They identified 1686 (1.3%) CAAs in a total of 126,595 patients who underwent coronary angiography at the Cleveland Clinic Foundation from 1960 to 1988. Separate origin of LAD and CX from the left SoV was the most seen anomaly with an incidence of 0.41% (n=513, study population=126,595). LAD and CX arise from separate ostia but adjacent from left SoV and their course and distribution are normal otherwise. This anomaly is benign in nature and has no hemodynamics compromise. Cannulation of the ostia during conventional coronary angiography may be difficult or even impossible in some patients; that could be misinterpreted as total obstruction or congenital absence of the vessels. Absence of LMCA trunk or

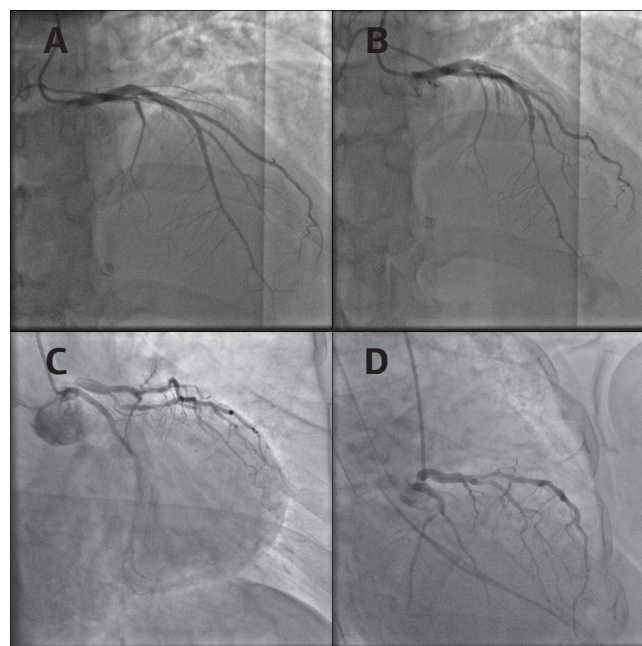


Figure 1. Coronary angiography images of muscular bridge in LAD artery (A-B). Coronary angiography images of two different patients with absent LMCA (C-D).

separate origin of LAD and CX from the left SoV is the second most seen anomaly in our study with a prevalence of 0.80% (n=17, study population=1617) and accounts for 23.28% (n=17, patients with CAAs=73) of CAAs. Our study has a slightly higher prevalence of absent LMCA than did Yamanaka O and Hobbs RE [10] and two previously published studies from our country with 0.23% and 0.64% [28, 29].

Coronary artery fistulas (Figure 2: A).

Coronary artery fistulas (CAFs), termination anomalies of coronary arteries, are abnormal connections between coronary arteries and any other structures. The incidence of CAFs was 0.18% (n=225, study population=126.595) and 13% (n=225, CAAs=513) as a proportion of CAAs in the study conducted by Yamanaka O and Hobbs RE [10]. Most of the CAFs arise from the LAD and RCA and drain off to right ventricle, right atrium, coronary sinus, pulmonary artery, left atrium and left ventricle. Myocardial ischemia, coronary steal, pulmonary arterial hypertension, congestive heart failure, bacterial endocarditis, and even rupture are among the presentations of CAFs. CAFs are the third most commonly seen anomalies in our study with a prevalence of 0.61% (n=10, study population=1617), accounting for 13.69% (n=10, patients with CAAs=73) of CAAs. CAFs prevalence varies from 0.11% [28] to 0.2% [29] in previously published studies from our country. Our findings are slightly higher than in these studies and we postulate that there could be variations from region to region and country to country when the subject is CAAs.

Origin of CX from right sinus of Valsalva (Figure 2: B).

In this anomaly CX arises from the right SoV, courses posterior to the aorta, and provides branches to the left lateral wall of the heart. It is a quite common anomaly. Yamanaka O and Hobbs RE [10] reported an incidence of 0.37% (n=467, study population=126.595) as the second most commonly seen anomaly

in their study. It is thought to be benign. It can be suspected when there is absent LMCA with LAD origination alone from the left SoV and nonperfused left lateral wall. It can be visualized by chance while trying to cannulate RCA. It can lead to increased radiation exposure to the operator or misinterpreted to be occluded during diagnostic procedures. It should be carefully reported in the patient's data and the surgeon must be alerted in case of cardiac surgeries as for all the other CAAs. We defined three patients out of 1617 patients with this anomaly, for a prevalence of 0.18%. Ten patients were identified to have anomalous origin of CX arising from right SoV out of 5548 patients who underwent coronary angiography, for a prevalence of 0.18% in a study conducted in our country [29]. Our data is compatible with the current literature in relation to anomalous origin of CX arising from right SoV.

Origin of RCA from left sinus of Valsalva (Figure 2: C).

Anomalous origin of RCA from left SoV is suspected when selective coronary angiography of RCA is unable to be performed from the right SoV. In this anomaly RCA originates from an orifice located anterior to the LMCA. It is very hard to cannulate this anomaly during selective coronary angiography due to its orifice and angulation. It arises from the left SoV and passes through the pulmonary artery and aorta. Angina pectoris, myocardial infarction, ventricular tachycardia, syncope, and sudden death can result from this anomaly even in the absence of coronary atherosclerosis. Yamanaka O and Hobbs RE [10] reported its prevalence as 0.107% (n=136, study population=126.595) in their study. We have a prevalence of 0.12% (n=2, study population=1617) in our present study. Altin et al. presented an incidence of 0.072 of anomalous origin of RCA from left SoV in studies from our country [29]. Our data is compatible with the current literature.

Absent Obtus Marginalis (OM)

There were no publications about absent OM in the literature. We came across this anomaly in two patients in our series. Diagonal branches of LAD supplied blood flow to the absent OM's area in these patients. The prevalence of the anomaly is 0.12 (n=2, study population=1617). To the best of our knowledge, ours is the first study to note the presence of this anomaly.

Woven coronary anomaly

Woven coronary anomaly, poorly understood to date, is division of an epicardial coronary artery into small branches: twisting of these small branches around their structure and merging again distally to form same coronary artery to supply myocardium. Transverse cut view of the vessel shows multiple small vessels in the area. Distal blood flow rate is generally preserved but may be compromised in some patients. Woven coronary anomaly is generally considered a benign anomaly. However, coronary artery dissection, thrombus formation and chronic total occlusion of artery with bridging collaterals could be confused as an alternative diagnosis and further unnecessary intervention could be performed. The incidence of the anomaly is not known. There are 23 adult cases published in the literature. RCA is the most affected artery followed by LAD and CX in this report [30]. The prevalence of this anomaly is 0.12 (n=2, study

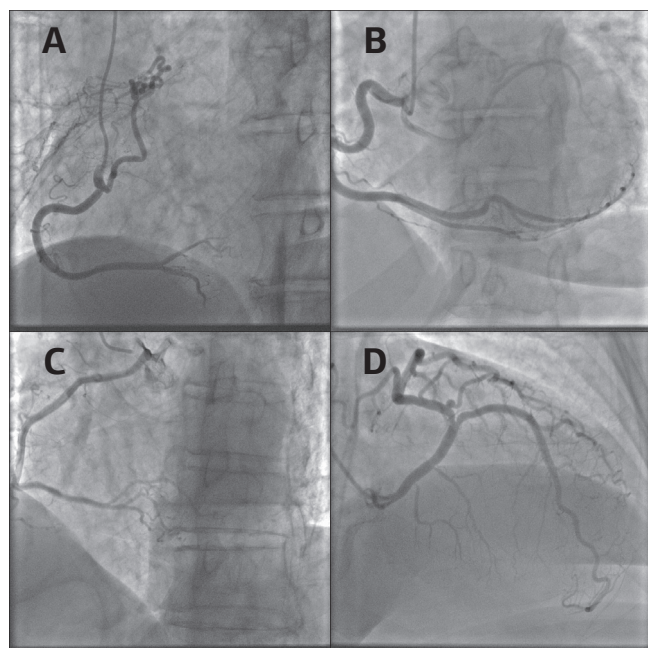


Figure 2. Coronary angiography image of coronary fistula arising from RCA (A). Coronary angiography image of CX arising from right anterior sinus(B). Coronary angiography image of RCA arising from left anterior sinus (C). Coronary angiography image of LMCA arising from right anterior sinus (D).

population=1617) in the present study. We had one patient who presented with acute inferior wall ST-elevation myocardial infarction (thrombotic occlusion of RCA) and Woven CAA in LAD. The other patient had Woven CAA in IM artery and OM at the same time. Distal blood flow was TIMI-3 (thrombolysis in myocardial infarction) in both patients.

LMCA arising from right sinus of Valsalva (Figure-2: D).

In this anomaly all the blood supplies of the left ventricle come from the right SoV via LMCA. There are some subtypes of this anomaly according to the origination and course of the LMCA to the aorta and pulmonary artery. "Anterior", "posterior", "septal", "between", and "combined" are the defined types of LMCA arising from right SoV. This anomaly is associated with increased risk of sudden cardiac death among patients without symptoms. The "between" type, in which LMCA arises from the right SoV and course through the aorta and pulmonary artery, is the most serious type. It is postulated that acute expansion of the aorta during exercise compromises the orifice of LMCA and leads to angina pectoris, syncope, myocardial infarction, ventricular tachycardia, or sudden cardiac death even in the absence of coronary atherosclerosis [31-33]. Yamanaka O and Hobbs RE [11] reported an incidence of 0.017% (n=22, study population=126,595) in their study. We identified one patient with LMCA arising from right SoV out of a total of 1617 with a prevalence of 0.061%. This anomaly was reported to have a prevalence of 0.029 (n=17, study population=58,023) in a previously published study from our country [28]. Our prevalence was slightly higher than in previously published studies.

RCA arising from posterior sinus of Valsalva

This anomaly is extremely rare. Yamanaka O and Hobbs RE [10] reported an incidence of 0.003% (n=4, study population=126,595). It is believed to be benign. In this anomaly the course of the vessel is normal. We identified one patient with RCA arising from posterior SoV out of a total 1617 coronary angiographies with a prevalence of 0.061% (n=1, study population=1617).

PDA arising from right anterior sinus of Valsalva

There were no publications about absent PDA arising from right anterior sinus of Valsalva in the literature. We came across this anomaly in one patient in our series. The prevalence of the anomaly is 0.061%. (n=1, study population=1617). Ours is the first study to claim this anomaly to our knowledge.

Single coronary artery (Figure 3: A).

Single coronary artery was first defined in 1941 and shown with angiography in two patients in 1967 [34]. This is a very rare coronary anomaly, generally associated with complex congenital heart defects, which makes it rare even among the elderly. All the myocardium is supplied with a single coronary artery in this anomaly. Clinical presentation depends on the course of the vessel. Sudden cardiac death and acute myocardial infarction was defined in patients with LMCA and/or RCA passing between the aorta and pulmonary artery during exercise. The incidence of sudden cardiac death is higher among anomalous origin of single coronary artery from right SoV. The prevalence

of anomaly is reported to be 0.02% to 0.04% in coronary angiography series. We identified one patient with single coronary artery arising from left SoV with a prevalence of 0.061% (n=1, study population=1617).

Dual LAD (Figure 3: B).

The presence of two coronary arteries in the anterior interventricular groove is identified as dual LAD. In this anomaly two vessels supply the septum and anterior wall of the left ventricle. Six subtypes of this anomaly are defined so far. In the first three forms the short and the long LADs arise from the LMCA. A short LAD originates from the LMCA and a long LAD from RCA in the fourth subtype. In the fifth subtype, the short LAD arises from the left SoV and the long LAD arises from the right SoV with an intramyocardial course. In the sixth subtype, the short LAD arises from the LMCA and the long one from the RCA with a course underneath right ventricular outflow tract. The anomaly is generally thought to be benign. The prevalence of the disease was reported to be 0.041% in 12,059 patients undergoing coronary angiography from the western side of Turkey [7]. We defined one patient with dual LAD with prevalence of 0.061% (n=1, study population=1617). In our patient both the short and the long LAD arose from the LMCA.

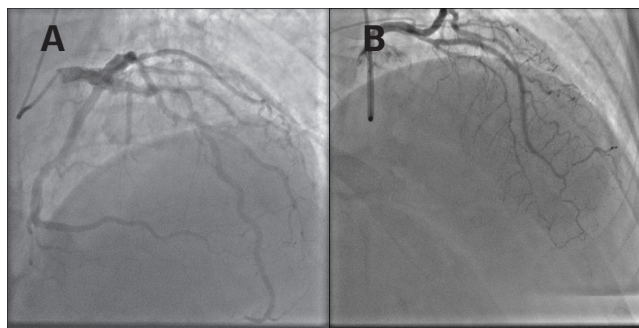


Figure 3. Coronary angiography image of single coronary artery (A). Coronary angiography image of dual LAD (B).

Coronary artery crossing

It is assumed that coronary arteries course parallel to each other and do not cross each other. It is a very rare anomaly and believed to be benign [35]. Despite coronary artery crossing being found usually in OM branches of CX, it can be found in LAD and RCA also. We defined one patient with coronary artery crossing in OM with a prevalence of 0.061% (n=1, study population=1617).

Selection of the patients to the study is the major limitation of our study as previously stated in the literature. The real world incidence of CAAs could not be determined properly because of the limited number of patients and biased inclusion criteria. Also, the fact that patients underwent coronary angiography can distort the incidence and prevalence because of their high probability of having coronary artery disease, ischemia and symptoms. The definition of CAAs varies from study to study (e.g., myocardial bridge is not accepted as a CAA in one of the milestone studies of CAA conducted by Yamanaka et al. with 126,595 patients). We are a tertiary center university hospital and we perform angiographies through femoral access site due to failure of radial access site or other issues. This can also be

a limitation to our study. Due to ethical concerns, the CAA diagnoses could not be confirmed with other imaging modalities (radiation exposure, lack of MRI angiography).

To date, there was no data concerning the prevalence of CAAs in the Black Sea Region. This is the first study performed in this area and the second one that showed the feasibility and safety of trans radial access in diagnosis of CAA. The incidence of CAA ranges from study to study according to definitions and geographical location. Some of the CAAs may be related to anginal symptoms, myocardial infarction, and sudden death despite generally being in benign nature. To avoid misunderstandings one must know the normal anatomy of coronary vasculature, variations and anomalies. When coronary angioplasty or cardiac surgery is planned in patients with CAA, special attention must be paid not to harm coronary arteries in unexpected locations.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding

None

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References

- Angelini P. Normal and anomalous coronary arteries: definitions and classifications. *American Heart Journal*. 1989; 117: 418-34
- Alexander RW, Griffith GC. Anomalies of the coronary arteries and their clinical significance. *Circulation*. 1956;14:800-5.
- Angelini P, Villason S, Chan AV, Diz JG. Normal and anomalous coronary arteries in humans. In: Angelini P. *Coronary artery anomalies: a comprehensive approach*. Philadelphia: Lippincott Williams & Wilkins. 1999; 27-150.
- Tuncer C, Batyraliev T, Yilmaz R, Gokce M, Eryoncu B, Koroglu S. Origin and distribution anomalies of the left anterior descending artery in 70,850 adult patients: multicenter data collection. *Catheter Cardiovasc Interv*. 2006; 68:574-85.
- Cademartiri F, La Grutta L, Malagò R, Alberghina F, Meijboom WB, Pugliese F et al. Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. *Eur Radiol*. 2008; 18:781-91.
- Gol MK, Ozturk MA, Kunt A, İçsan Z, Ozatik MA, Yavas S et al. Coronary artery anomalies in adult patients. *Med Sci Monit*. 2002; 8: 636-41.
- Aydinlar A, Çiçek D, Şentürk T, Gemici K, Serdar OA, Kazazoglu AR et al. Primary congenital anomalies of coronary arteries: A coronary arteriographic study in Western Turkey. *Int Heart J*. 2005; 46 : 97-103.
- Aydar Y, Yazıcı HU, Birdane A. Gender Differences in the Types and Frequency of Coronary Artery Anomalies. *Tohoku J. Exp. Med*. 2011; 225: 239-47.
- Özbek K, Katlandur, Keser HA. Screening of coronary artery anomalies in 11,707 patients reveals that the radial approach is safe for cannulating coronary anomalies. *European Review for Medical and Pharmacological Sciences*. 2016; 20: 1161-7.

- Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. *Cathet Cardiovasc Diagn*. 1990; 21:28-40.
- Altun B, Arici M, Nergizoglu G, Derici U, Karatan O, Turgan C et al. Prevalence awareness, treatment and control of hypertension in Turkey (the PatenT study) in 2003. *J Hypertens*. 2005; 23:1817-23.
- Satman I, Yilmaz T, Sengül A, Salman S, Salman F, Uygur S et al. Population-based study of diabetes and risk characteristics in Turkey. *Diabetes care*. 2002;25:1551-6.
- Harikrishnan S, Jacob SP, Tharakan J, Titus T, Kumar VK, Bhat A, et al. Congenital coronary anomalies of origin and distribution in adults: a coronary arteriographic study. *Indian Heart J*. 2002;54:271-5.
- de Jonge GJ, van Ooijen PM, Piers LH, Dikkers R, Tio RA, Willems TP, et al. Visualization of anomalous coronary arteries on dual source computed tomography. *Eur Radiol*. 2008;18:2425-32.
- Bestetti RB, Costa RS, Kazava DK, Oliveira JS. Can isolated myocardial bridging of the left anterior descending coronary artery be associated with sudden death during exercise? *Acta Cardiol*. 1991;46:27-30.
- den Dulk K, Brugada P, Braat S, Hedde B, Wellens HJ. Myocardial bridging as a cause of paroxysmal atrioventricular block. *J Am Coll Cardiol*. 1983;1:965-9.
- Marchionni N, Chechi T, Falai M, Margheri M, Fumagalli S. Myocardial stunning associated with a myocardial bridge. *Int J Cardiol*. 2002;82:65-7.
- Feld H, Guadanino V, Hollander G, Greengart A, Lichstein E, Shani J. Exercise-induced ventricular tachycardia in association with a myocardial bridge. *Chest*. 1991;99:1295-6.
- Tio RA, Ebels T. Ventricular septal rupture caused by myocardial bridging. *Ann Thorac Surg*. 2001;72:1369-70.
- Berry JF, von Mering GO, Schmalfuss C, Hill JA, Kerensky RA. Systolic compression of the left anterior descending coronary artery: a case series, review of the literature, and therapeutic options including stenting. *Catheter Cardiovasc Interv*. 2002;56:58-63.
- Gowda RM, Khan IA, Ansari AW, Cohen RA. Acute ST segment elevation myocardial infarction from myocardial bridging of left anterior descending coronary artery. *Int J Cardiol*. 2003;90:117-8
- Rossi L, Dander B, Nidasio GP, Arbustini E, Paris B, Vassanelli C et al. Myocardial bridges and ischemic heart disease. *Eur Heart J*. 1980;1:239-45.
- Geiringer E. The mural coronary. *Am Heart*. 1951;41:359-68.
- Schwarz ER, Klues HG, vom Dahl J, Klein I, Krebs W, Hanrath P. Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication. *J Am Coll Cardiol*. 1996;27:1637-45.
- Ge J, Jeremias A, Rupp A, Abels M, Baumgart D, Liu F et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. *Eur Heart J*. 1999;20:1707-16.
- Gertz SD, Uretsky G, Wajnberg RS, Navot N, Gotsman MS. Endothelial cell damage and thrombus formation after partial arterial constriction: relevance to the role of coronary artery spasm in the pathogenesis of myocardial infarction. *Circulation*. 1981;63:476-86.
- Ziegler T, Bouzourene K, Harrison VJ, Brunner HR, Hayoz D. Influence of oscillatory and unidirectional flow environments on the expression of endothelin and nitric oxide synthase in cultured endothelial cells. *Arterioscler Thromb Vasc Biol*. 1998;18:686-92
- Turkmen S, Cagliyan CE, Poyraz F, Sercelik A, Boduroglu Y, Akilli RE, et al. Coronary arterial anomalies in a large group of patients undergoing coronary angiography in southeast Turkey. *Folia morphologica*. 2013;72:123-7
- Altin C, Kanyilmaz S, Koc S, Gursay YC, Bal U, Aydinalp A, et al. Coronary anatomy, anatomic variations and anomalies: a retrospective coronary angiography study. *Singapore medical journal*. 2015 Jun;56(6):339
- Val-Bernal JF, Malaxetxebarria S, González-Rodilla I, Salas-García M. Woven coronary artery anomaly presenting as sudden cardiac death. *Cardiovascular Pathology*. 2017 Feb 28;26:7-11
- Kragel AH, Roberts WC: Anomalous origin of either the right or left main coronary artery from the aorta with subsequent coursing between aorta and pulmonary trunk: analysis of 32 necropsy cases. *Am J Cardiol*. 1988;62:771-7.
- Liberthson RR, Dinsmore RE, Bharati S, Rubenstein JJ, Caulfield J, Wheeler EO, et al: Aberrant coronary artery origin from the aorta: diagnosis and clinical significance. *Circulation*. 1974;50:774-9.
- Halperin IC, Penny JL, Kennedy RJ. Single coronary artery. Antemortem diagnosis in a patient with congestive heart failure. *Am J Cardiol*. 1967; 19: 424-7.
- Toshima H, Sasaki K, Oshima F, Tanaka K, Fukami T. Single coronary artery. Case report. *Kurume Med J*. 1967; 14: 89-93.
- Kursaklıoğlu H, İyisoy A, Çelik T, Günay C. Koroner Arter Anomalileri. In: *Koroner arter anomalileri*. Ali Oto, Hürkan Kuşaklıoğlu, Atilla İyisoy (eds). Koroner arter anomalileri. Hacettepe Ün. Hastaneleri basımevi, birinci basım. 2005, 16-91.

How to cite this article:

Kayapınar O, Sayın AE, Kaya A, Özde C, Keskin M. Prevalence and characteristics of CAAs in the black sea region. *J Clin Anal Med* 2018;9(5): 369-75.